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CC-chemokine Co-receptor R5 genotypes in Ghanaian couples discordant for human immunodeficiency type 1 infection

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Background: The prevalence of HIV among heterosexual couples is high. In spite of this, there are instances in which one partner is HIV sero-positive and the other HIV sero-negative (described as discordant couple) despite being at risk for HIV infection. A number of factors are responsible among which is a deletion in the major coreceptor (chemokine receptor CCR5) for entry of HIV-1 into CD4+ T lymphocyte cells. Individuals homozygote for a 32 base pair deletion (CCR5 Delta 32) do not express the CCR5 receptor at their cell-surface and have a natural resistance to HIV-1 infection.

Methods: In order to determine the CCR5 genotypes of Ghanaian serologically discordant couples and the possible role of CCR5 Delta32 mutant allele in the lack of HIV-1 transmission, 32 couples (serologically discordant couples SDC and serologically concordant couples SCC) visiting the Fevers Unit (FU) of the Korle-Bu Teaching Hospital (KBTH) were enrolled. Couples were made to fill questionnaires on their behavioural characteristics and blood samples were taken for HIV-1 antibody testing and confirmation. HIV-1 negative serostatus of discordant partners was confirmed by polymerase chain reaction (PCR). The CCR5 genotypes were determined using DNA subjected to PCR amplification.

Results: Couples response to questionnaires revealed that they indulged in high risk behavioural practises.HIV antibody testing with PCR confirmation revealed 8 SDC and 24 SCC. One SDC seronegative individual (out of 8 SDC seronegatives) was heterozygous for the CCR5 Delta32 genotype. This was an important finding (although possession of CCR5 Delta32 heterozygous genotype may delay the acquisition of HIV-1 infection but cannot be implicated in natural resistance to HIV-1 infection) because most research scientist augment that the gene mutation does not exist in people of African decent. This was the first time that the CCR5 genotypes of Ghanaians were determined and also the first time that a heterozygous was found. The rest of the couples had wild type CCR5 genotype.

Conclusion: CCR5 Delta 32 mutant allele is unlikely to explain discordance in this cohort. There the need for a population based study to help ascertain the prevalence of CCR5 genotypes in the Ghanaian population. Counselling and follow-up must also be intensified in the group at risk group.

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Endothelial Progenitors Cells and Microparticles in HIV infected patients

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Background: HIV infection is an inflammatory disease and can cause endothelial disfunction and increases the cardiovascular risk. It plays a critical role in coronary artery disease, but the assessment of the endothelial disfunction has been problematical. Exposure to cardiovascular risk factors alters the regulatory functions of the endothelium that progresses from a quiescent state to activation, apoptosis and death. Recently, it has been shown that endothelial cells (EC) release microparticles (MP) on activation or apoptosis and that evaluation of MP can provide useful information on EC status in patients with an increased cardiovascular risk. Identification of circulating endothelial cells and microparticles has raised considerable interest as non-invasive markers of vascular disfunction.

Methods: We studied these new markers of cardiovascular risk, as endothelial progenitors cells and microparticles in HIV-infected naive patients and compared with HIV negative controls matched for age and sex.Standard laboratory study included: lipid profile, glycaemia, C-reactive protein and apolipoprotein B. The endothelial progenitors (EPC) cells and microparticles (MP) were measure by flow citometry. The EPC were identified using the following markers: CD34+ (surface protein), KDR (vascular endothelial growth receptor 2) for definition and CD133+ for immature lineage cells. The MP were characterized with CD31+ (MP platelet and endothelial derived), CD42+ (MP platelet derived) and CD51+ (MP endothelial derived).

Results: Thirty patients were included, 15 in each group, 73,3% were male with mean age 30,9 years. The lipid profile was significant only in the HDL-c and LDL-c between the groups. In the HIV-infected group we observed 0,01% of CD34+/KDR+ and it was not isolated CD34+/CD133+ neither CD133+/KDR+. In this group it was also observed more release of MP CD51+ and CD31+/CD42+ comparing to the control group, any MP CD31+/CD42- were found.

Conclusion: Our results suggest a possible imbalance between EPC and MP. This new finding in HIV-naive patient may be associated with increased cardiovascular disease in the long term follow-up of these patients, and can be aggravated after antiretroviral therapy.

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